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REVIEW

Lichen planus is associated with hepatitis C but not with hepatitis B virus: a systematic review and meta-analysis with a focus on Italian data

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ABSTRACT

BACKGROUND: Several studies have explored the potential association between lichen planus (LP) and chronic hepatitis C (HCV) and hepatitis B virus (HBV) related, yielding conflicting results. Objective: To determine whether there is an association between these two entities, through a systemic review and meta-analysis.
METHODS: Bibliographic searches were conducted using PubMed and Scopus databases. Data obtained from papers included in the systematic review were put into a quantitative meta-analysis, carried out with SPSS software version (29.0.2.0).
RESULTS: Out of the initial pool of 834 studies, 45 studies met the inclusion criteria for meta-analysis. The odds ratio (OR) for HCV-seropositivity in LP patients was 4.55 (95% CI, 3.08-6.74), with higher ORs observed in Mediterranean basin countries (OR: 5.41, 95% CI: 3.16-9.29). In Italy, the OR was 4.42 (95% CI, 1.99-9.81). A regional variation was noted within Italy, with a higher OR in northern Italy-compared to southern Italy. Similar associations were found in Asian countries, with an OR of 4.49 (95% CI, 2.94-6.88). However, in northern Europe, the pooled OR was 0.733 (95% CI, 0.15-3.66), indicating no statistically significant association. When considering subjects with HCV infection, the presence of LP showed a pooled OR of 3.22 (95% CI, 1-10.34). Regarding HBV infection, the OR was 1.51 (95% CI, 1.15-1.97), with no differences and was similar when analyzing different subgroups.
CONCLUSIONS: Our study indicates a moderate to high risk for HCV in patients with LP, according to geographic region, while the risk remains uncertain for HBV with respective ORs of 4.55 (95% CI, 3.08-6.74) and 1.51 (95% CI, 1.15-1.97). It seems reasonable to test the sera of patients affected by LP for anti-HCV antibodies, while the necessity of testing for HBV remains more uncertain.

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KEY WORDS: Lichen planus; Hepatitis C; Hepatitis B.

Lichen planus (LP) is a chronic mucocutaneous inflammatory disease, affecting between 0.14% and 1.27% of the general population.[1] LP typically affects middle-aged adults, with a slight predilection for women.[2]

LP involves the stratified squamous epithelium, especially the skin and oral mucosa, but other mucous membranes (genitalia, esophagus, conjunctiva) and skin appendages (scalp hair, nails) can be involved.[3]

The exact cause of LP is not fully understood, but it is considered a manifestation of cell-mediated immune response.[4, 5] Histologically, LP is characterized by a dense subepidermal lymphocytic infiltrate, mostly made by CD8+ T cells.[1] Langerhans cells and macrophages are increased too and provide antigenic signals for autoreactive CD8+ T-cell activity, directed against the epithelial basal layer. This immune response results in the liquefaction of the basal layer, apoptosis of basal keratinocytes, acanthosis, thickening of the granular cell layer and often hyperkeratosis.[6]

Several theories suggest that Hepatitis C Virus (HCV) may trigger an innate and adaptive immune response contributing to LP pathogenesis. The first cases of oral lichen planus (OLP) potentially linked to HCV infection were reported in the 1980s-90s by Rebora and Rongioletti.[7, 8]

Subsequently, numerous investigations have explored the relationship between HCV and OLP across various countries. However, these studies have yielded contradictory findings, with some indicating a positive correlation, while others have documented a lack of association.[9]

Additional investigations into the relationship between LP and Hepatitis B Virus (HBV), while less robust compared to HCV, did not provide any pathogenetic mechanism and showed debatable results, to date.[10]

According to this evidence, this field remains to be explored, and further investigations are required to elucidate mechanisms underlying these potential associations. Therefore, the aim of this paper is to provide a systematic review and meta-analysis to explore the relationship between LP and viral infections, particularly HCV and HBV, taking into account geographical variations and a specific focus on data reported in Italy.

Materials and methods

Search strategies

A web-based search of the available literature was carried out by physicians using the databases of PubMed and Scopus. At first, the combined search terms "lichen planus AND HCV" were used. Subsequently, the focus shifted to HBV, using the terms "lichen planus AND HBV." Our search included studies from inception to November 2024.

Selection process

The screening procedure adhered to the guidelines outlined by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA).

For all studies identified through the described search strategies, the abstract was examined. Studies investigating the association between LP and hepatitis B and C virus were selected independently (S.G., M.T.).

After this initial screening, full-text studies were examined. The eligibility criteria for selecting studies were as follows:

- study design: studies employing appropriate research designs to investigate the relationship between lichen planus and HCV, such as case-control or cross-sectional studies;
- diagnostic methods: studies utilizing validated and standardized diagnostic criteria for lichen planus and HCV infection. Histological confirmation was deemed necessary for the diagnosis of LP;
- matching of the study group and control group for factors such as age and gender;
- minimum case threshold: studies including a minimum of 15 cases were included in the analysis;
- outcomes: studies reporting clear and detailed results on the association between lichen planus and HCV, allowing the calculation of measures of association such as odds ratio.

Data collection process

Data pertinent to the investigation were meticulously extracted from each individual study, encompassing essential details such as the primary author's identity, year of publication, study design, geographical location, distinctive attributes of both case and control cohorts, as well as the diagnostic methodology employed therein.

Statistical analysis

SPSS software version 29.0.2.0 (Armonk, NY, USA) was used for statistical analysis. Pooled proportions with 95% confidence intervals (CIs) were calculated. The weighted summary proportion under the fixed effects model was computed with Mantel-Haenszel transformation in the absence of heterogeneity. The random-effects model was used when heterogeneity was moderate, large, or extreme. Pooled proportions (±95% CI) and the overall effect (±95% CI) were visualized using forest plots. The size of the marker is relative to the weights assigned to the different studies. Pooled effects are displayed as diamonds, where its location represents the estimated effect size, and its precision is reflected by its width.

Heterogeneity between studies was assessed using the χ^2-based Cochran's Q statistic test and I^2 metric. Heterogeneity was considered significant at $P<0.01$ for the Q statistic (if the observed variance exceeds the expected variance), and I^2 metric ($I^2 = 100\% \times (Q-df)/Q$) uses cut-off points of no heterogeneity (I^2: 0-25%), moderate (I^2: 25-50%), large (I^2: 50–75%) and extreme (I^2: 75-100%) heterogeneity.

Results

Study selection

A total of 834 records were identified with the initial search. Overall, 352 duplicates and two studies written in languages other than English were excluded.

For all studies identified through the described search strategies, the abstract was examined; 252 studies were excluded based on the title and abstract assessments, 228 studies were available for full-text assessment.

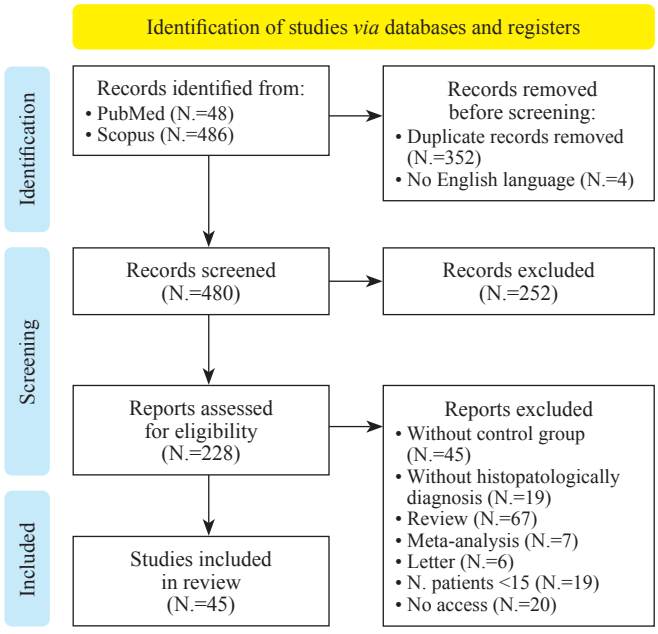

Figure 1.—PRISMA diagram flow.

Ultimately, out of the initial pool of 834 studies, a total of 45 studies[5, 11-54] met the criteria for inclusion in the meta-analysis (Figure 1).

Study population

The total number of studies for determining HCV in LP included 4380 LP patients who were 618 HCV+, and 231423 individuals served as a control group, with 520 HCV+ subjects (Supplementary Digital Material 1: Supplementary Table I).[5, 11-50]

The total number of studies for determining HBV in LP included 1351 LP patients who were 146 HBV+, and 1700 individuals served as a control group, with 118 HBV+ subjects (Table I).[51-54]

Oral localization is present in the majority of studies, absent only in three studies. Cutaneous localization, on the other hand, was absent in 17 studies.

Additionally, four studies investigated the presence of LP in HCV infected patients were analyzed.

HCV seropositivity in LP patients

In forty-one studies LP patients and control subjects were investigated for HCV-seropositivity.[5, 11-50]

Three studies were included twice in the meta-analysis because the authors included two different control groups. In eleven studies, no seropositive patients were found in either group. The OR of HCV seropositivity in patients with LP varied between 0.23 (95% CI, 0.01-5.85) and 47.71 (95% CI, 14.86-153.26). The pooled OR acquired was OR 4.55 (95% CI, 3.08-6.74), indicating a statistically significant difference in the proportion of HCV-seropositivity subjects among patients with LP, compared to controls. The effect size was 1.516 and the heterogeneity between the studies was assessed as large, with an I^2 of 64.8% (Figure 2).

Geographic variability in HCV seropositivity in LP patients

Sub-analyses were carried out taking into account geographical variability. Studies were grouped into three main macro-areas: the Mediterranean basin, Asian Countries and Northern Europe. The Mediterranean basin included Italy, Türkiye, Spain, Egypt, Israel. Asian countries included India, Thailand, Japan, China, Pakistan, Iran, Saudi Arabia and Nepal. Northern Europe included the UK and Germany.

In Mediterranean basin[5, 12, 16, 19, 22, 23, 25, 26, 29, 33, 43, 50] the pooled OR was 5.41 (95% CI, 3.16–9.29), suggesting a strong risk of HCV in LP patients. The effect size was 1.689 and the I^2 was 72%.

Among Mediterranean basin, a further sub-analysis was conducted considering Italy,[5, 13, 23, 33, 35] where the pooled OR acquired was 4.42 (95% CI, 1.99-9.81). The effect size was 1.486 and the I^2 was 84% (Figure 3). It is important, however, to underline that within the same Country, there were differences between regions. For instance, within Italy, for Northern regions[33, 35] an OR of 7.35 (95% CI, 3.92-13.79), with an effect size of 1.995, was estimated while in Southern Italy[5, 13, 23] it was 3.32 (95% CI, 1.04-10.59), with an effect size of 1.2. The heterogeneity was observed only in studies of Southern Italy, with a value of 88%.

In Asian areas[15, 17, 18, 21, 24, 30, 31, 34, 36, 37, 40, 45, 48] the pooled OR calculated was 4.49 (95% CI, 2.94-6.88), a value fairly

TABLE I.—*Studies investigating the presence of LP in HCV infected patients.*

Study	Study design	Country	HCV patients (total)	Mean age of HCV patients	Controls (total)	Mean age of control individuals
Cribier[51]	Case control, retrospective	France	100	48	50	51.46
Cunha[52]	Case control, retrospective	Brazil	134	51	95	45
Dervis[53]	Case control, retrospective	Türkiye	70	54.14	70	51.66
Maticic[54]	Case control, prospective	Slovenia	171	38	171	40

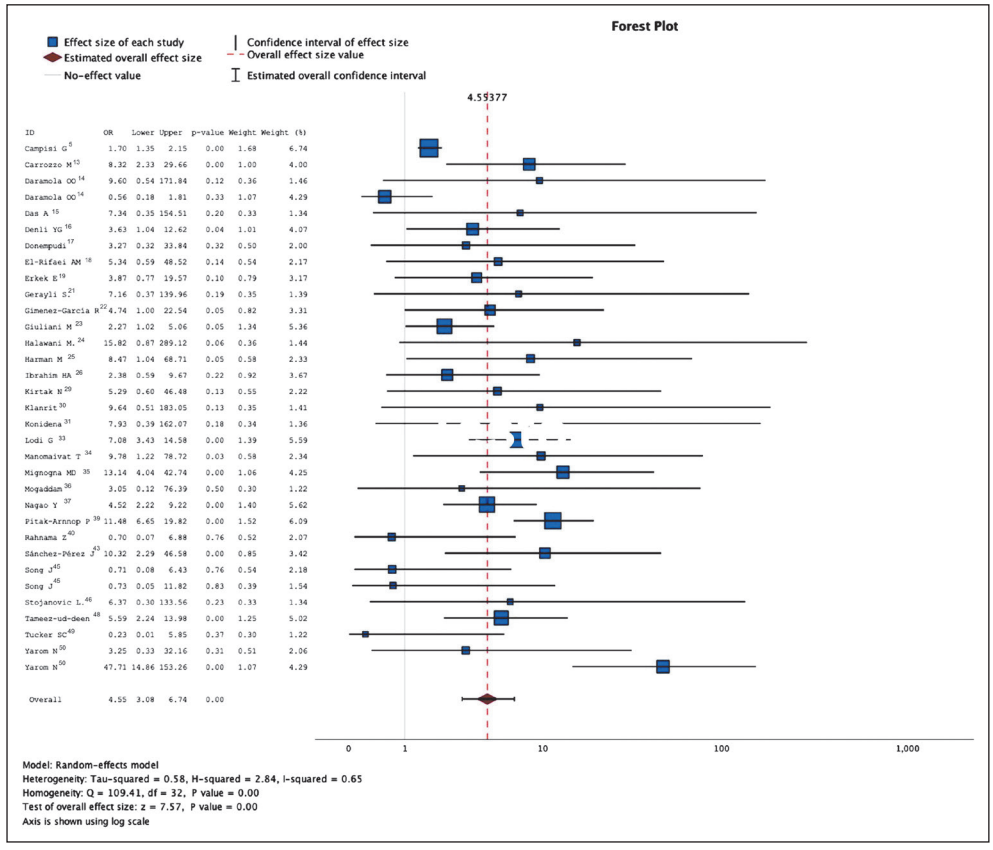

Figure 2.—Odds ratios and forest plots of all studies included relating hepatitis C infection to lichen planus.

consistent with the OR calculated across all studies. The effect size was 1.502 with no observed heterogeneity.

In Northern Europe[27, 42, 49] the pooled OR acquired suggested no statistically significant association. In this case it was necessary to correct zero values by adding 1 in two of the three studies, in order to calculate the OR. The final value was 0.73 (95% CI, 0.15–3.66), the effect size was -0.311 and no heterogeneity was observed.

Type of study, number of participants and clinical manifestations

Additional analyses were conducted to test LP and HCV correlation, based on the type of studies, number of participants and clinical manifestations.

Prospective studies[13, 17, 19, 40, 49] showed an OR of 2.77 (CI 95%, 0.92-8.35), with an effect size of 1.02 and an I^2 of 36%. So, considering prospective studies, the association appears to be lower as compared to overall. Retrospective studies[5, 14-16, 18, 21-26, 29-31, 33-37, 39, 43, 45, 46, 48, 50] showed an OR of 4.90 (CI 95%, 3.19-7.51). The effect size was 1.589 and the I^2 was 68%.

When considering only studies with more than 100 LP patients,[5, 15, 16, 21, 22, 25, 33-35, 39, 45, 46] the OR was 4.92 (CI 95%, 2.78-8.72), with an effect size of 1.593 and I^2 of 68%.

Studies limited to patients with oral manifestations,[5, 17, 18, 21, 23, 29, 34, 37, 45, 50] without cutaneous involvement, were selected. The OR obtained was 3.98 (95% CI, 1.93-8.18), with an effect size of 1.380 and an I^2 of 75%.

Presence of LP in HCV patients

In the studies investigating the presence of LP in HCV patients[51-54] the pooled OR acquired was 3.22 (95% CI, 1-10.34), thus supporting once again an association between the two diseases. The effect size was 1.17 and the I^2 was 4%.

HBV seropositivity in LP patients

The OR of HBV seropositivity in patients with LP[1-20, 24, 26, 28, 30, 32, 35, 36, 38, 43, 45] varied between 0.33 (95% CI, 0.01-8.21) and 3.21 (95% CI, 1.10-9.33). The pooled OR acquired was OR 1.51 (95% CI, 1.15-1.97) and the effect size was 0.409, with no observed heterogeneity (Figure 4).

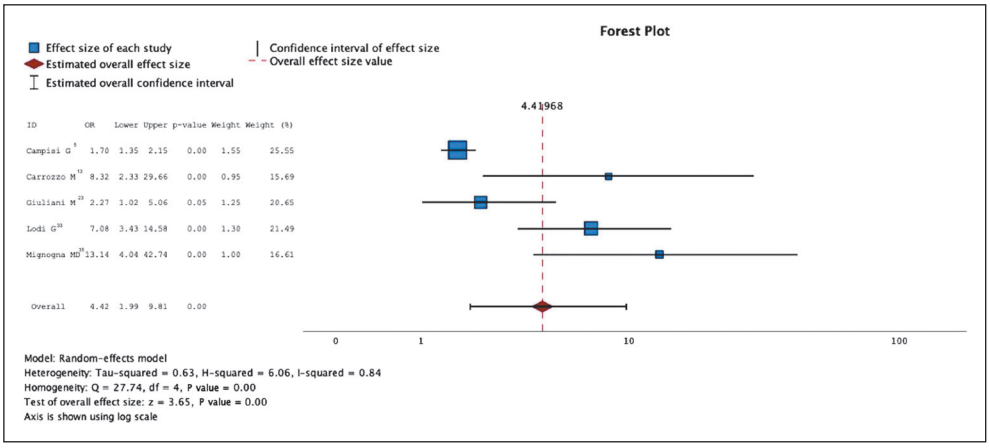

Figure 3.—Odds ratios and forest plots of all studies included concerning hepatitis C infection to lichen planus in Italy.

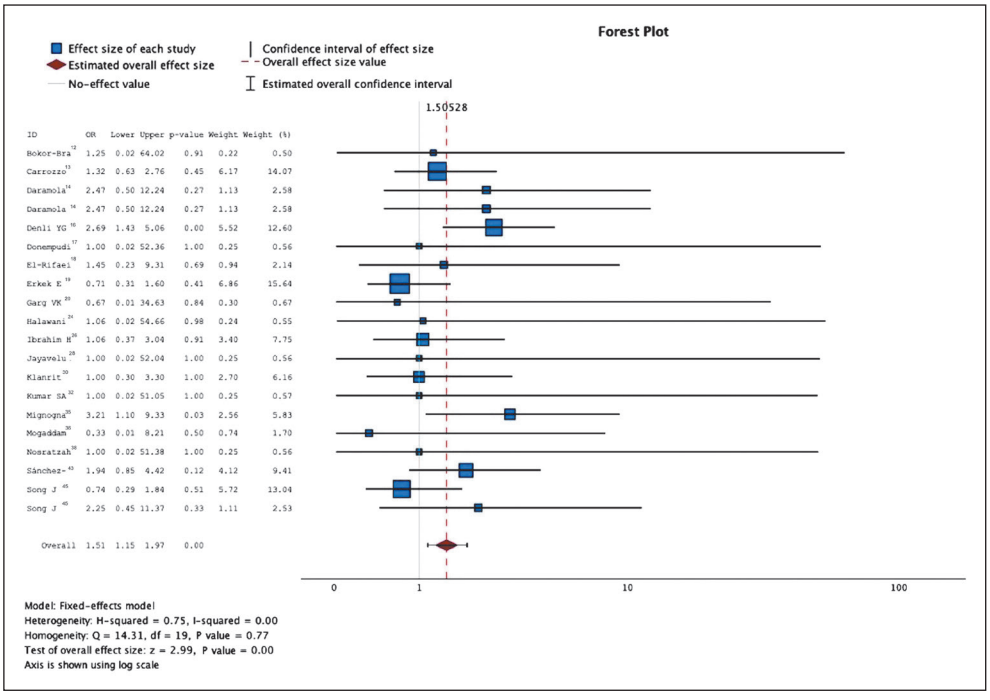

Figure 4.—Odds ratios and forest plots of all studies concerning HCV seropositivity in LP patients.

Since the highest OR was found in Italy, a separate analysis was performed,[13, 35] showing a value of 1.89 (95% CI, 0.81-4.41). The effect size was 0.635 and the I^2 was 44%.

In contrast, considering Asian countries,[18, 30, 36, 45] the OR was 0.98 (CI 95%, 0.55-1.77), with an effect size of -0.018 and no heterogeneity observed.

Among Mediterranean basin countries,[13, 16, 19, 26, 35, 36, 43, 45] the OR was consistent with the overall OR, with a value of 1.45 (CI 95%, 0.96-2.20), an effect size of 0.373 and an I^2 of 39%.

When prospective studies were considered,[13, 19] no significant association was found, with an OR of 1.00 (CI 95%, 0.58-1.72), an effect size of 0 and an I^2 of 20%.

When retrospective studies were considered[14, 16, 18, 26, 30, 35, 39, 43, 45] the OR was 1.76 (CI 95% 1.28-2.42), with an effect size of 0.567 and no observed heterogeneity.

When taking in consideration only studies with more than 100 LP patients,[16, 35, 45] the OR was 1.92 (CI 95%, 0.94-3.94). The effect size was 0.654 and the I^2 was 58%.

Studies limited to patients with oral manifestations, without cutaneous involvement,[18, 30, 45] showed no signifi-

cant association, with an OR of 1.03 (95% CI, 0.56-1.88). The effect size was 0.028 with no observed heterogeneity.

Discussion

This systematic review with meta-analysis shows that LP patients have an overall fourfold increased risk of HCV seropositivity. This risk has been confirmed also in HCV patients affected by LP, revealing a threefold increased risk.[53] The average prevalence of HCV in LP patients is 1.9%, while that of HBV is 0.9%.[55]

These results are consistent with those reported in other meta-analyses examining the relationship between HCV and LP, demonstrating an increased association. Additional sub-analyses including different geographic areas, type of studies, number of patients and location of LP were carried out, revealing interesting correlations.

Our results suggest that the relationship between LP and HCV is influenced by the geographic site not only between different continents but also within the same area or Country. Specifically, the risk of HCV in LP patients in the Mediterranean basin areas is fivefold, higher as compared to the overall risk. Instead, in Northern European countries like Germany and the UK there is no correlation. It is important to note that both Germany and the UK have low HCV infection rates, at 0.1% and 0.002%, respectively.[56] When prospective studies were considered, the OR was lower, with a value of 2.77 (95% CI, 0.92-8.35).

Interestingly, a regional variation was noted within Italy, with a higher ORs in Northern Italy, where it is 7.35 (95% CI, 3.92-13.79), as compared to Southern Italy, where it is 3.32 (95% CI: 1.63-2.51). A factor to consider is that in Southern Italy HCV is more frequent, but this alone may not be enough to explain such a difference.[57]

In addition, studies limited to patients with OLP without skin manifestations found a significant association with HCV with an OR of 3.98 (95% CI, 1.93-8.18).

The link between HCV and LP can be explained by the fact that HCV induces chronic inflammation, immune activation and antigenic changes.[58, 59] The significantly higher percentage of HCV-infected keratinocytes in LP lesions compared to healthy skin supports the hypothesis that HCV may contribute to the development of cutaneous LP through direct viral involvement or immune-mediated mechanisms.[60] Additionally, the resolution of LP following viral clearance through antiviral therapy supports a connection between the two entities.[61] Interferon also plays a role, as it has significant implications for therapy, although direct-acting antivirals appear to offer better resolution of HCV associated LP lesions, compared to interferon therapy.[62] However, genetic predispositions further influence this association, highlighting how various factors contribute to the link between the HCV and LP.[63]

Furthermore, our study investigated the association between the HBV and LP. An OR of 1.51 (95% CI, 1.15-1.97) for HBV has been observed in patients with LP. However, the risk is higher in Italy with a value of 1.89 (95% CI, 0.81-4.41), as compared to other geographical areas. Accordingly, Asian countries showed an OR of 0.98 (CI 95%, 0.55-1.77). Prospective studies revealed no significant association either, with an OR of 1.00 (CI 95%, 0.58-1.72). Additionally, studies limited to patients with oral manifestations, without cutaneous involvement showed no significant association with HBV, with an OR of 1.03 (95% CI, 0.56-1.88). No pathogenetic link has been provided to support the correlation between HBV an LP, therefore supporting the overall uncertain risk revealed with the current study.

Compared with the meta-analysis by María García-Pola *et al.*,[1] this study also analyzed the association with HBV and provided not only a detailed geographic analysis but also a focus on Italian data.

Conclusions

In conclusion, it seems reasonable to test the sera of patients affected by LP for anti-HCV antibodies, while it remains more uncertain whether to test patients with for HBV.

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Conflicts of interest
The authors certify that there is no conflict of interest with any financial organization regarding the material discussed in the manuscript.

Authors' contributions
Stefania Guida and Margherita Tamburelli contributed equally. All authors read and approved the final version of the manuscript.

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